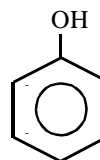


PHENOL

Phenol is a federal hazardous air pollutant and was identified as a toxic air contaminant in April 1993 under AB 2728.

CAS Registry Number: 108-95-2

Molecular Formula: C_6H_6O



Phenol is a white crystalline mass which turns red or pink if exposed to air or light. It has a burning taste and a distinct aromatic, acrid odor. It is very soluble in liquid sulfur dioxide, acetic acid, carbon tetrachloride and alcohol, and soluble in chloroform, ether, glycerol, petrolatum, carbon disulfide, volatile and fixed oils, aqueous alkali hydroxides, and acetone. It is slightly soluble in mineral oil. It is almost insoluble in petroleum ether. One gram of phenol dissolves in 15 milliliters (ml) of water and in 12 ml of benzene (Merck, 1989). Phenol is combustible when exposed to heat, flame, or oxidizers and emits toxic fumes when heated (Sax, 1989). It is incompatible with strong oxidizers and calcium hypochlorite (Sittig, 1985).

Physical Properties of Phenol

Synonyms: carbolic acid; phenic acid; phenylic acid; phenyl hydroxide; hydroxybenzene; oxybenzene; phenyl hydrate; monohydroxybenzene

Molecular Weight:	94.11
Boiling Point:	182.0 °C
Melting Point:	43.0 °C
Flash Point:	175.0 °F
Vapor Density:	3.24 (air = 1)
Density/Specific Gravity:	1.0576 at 20 °C (water = 1)
Vapor Pressure:	0.3513 mm Hg at 25 °C
Log Octanol/Water Partition Coefficient:	1.46
Conversion Factor:	1 ppm = 3.85 mg/m ³

(HSDB, 1991; Merck, 1989)

SOURCES AND EMISSIONS

A. Sources

Phenol is a common industrial chemical that is used in the manufacture of resins, plastics, fibers, adhesives, iron, steel, aluminum, leather, and rubber. It is also found in disinfectants, cleaners, cigarette smoke, and motor vehicle emissions (Howard, 1990; HSDB, 1991).

Phenol is used in a variety of indoor products. It is used in general disinfectants and as a biocide in paints. It is also used in medicinal preparations such as mouth washes, throat lozenges, and shave creams (Hodgson and Wooley, 1991). Environmental tobacco smoke is also a source of phenol.

Phenol was registered for use as a pesticide; however as of December 31, 1993, it is no longer registered for pesticidal use in California (DPR, 1996).

The primary stationary sources that have reported emissions of phenol in California are manufacturers of miscellaneous nonmetallic mineral products, petroleum refining, and paperboard mills (ARB, 1997b).

B. Emissions

The total emissions of phenol from stationary sources in California are estimated to be at least 510,000 pounds per year, based on data reported under the Air Toxics "Hot Spots" Program (AB 2588) (ARB, 1997b).

C. Natural Occurrence

Phenol is obtained from coal tar and is found in animal wastes and decomposition of organic wastes (HSDB, 1991).

AMBIENT CONCENTRATIONS

No Air Resources Board (ARB) data exist for ambient measurements of phenol. However, the United States Environmental Protection Agency (U.S. EPA) has compiled data from several urban locations throughout the United States from 1974-82. These data have an overall mean concentration of 0.03 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) or 0.008 parts per billion (U.S. EPA, 1993a).

INDOOR SOURCES AND CONCENTRATIONS

Potential indoor sources include building materials such as insulation, floor and wall coverings, wood-burning appliances, and cigarette smoke (Hodgson and Wooley, 1991).

A recent study conducted for the ARB measured emissions of six commercial cigarettes and one reference cigarette. Phenol had an average emission rate of 438 nanograms per milligram of tobacco or 281 micrograms per cigarette (Daisey et al., 1994).

No indoor concentration data for phenol have been reported for United States residences (Hodgson and Wooley, 1991).

ATMOSPHERIC PERSISTENCE

Phenol exists in the atmosphere predominantly in the gas phase. The dominant atmospheric loss processes for phenol are by daytime reaction with the hydroxyl (OH) radical and nighttime reaction with the NO₃ radical. The calculated half-life and lifetime of phenol due reaction with the OH radical are 9 hours and 13 hours, respectively. The products of the OH radical and NO₃ radical reactions include 2-nitrophenol (Atkinson, 1994). Phenol also undergoes wet deposition, but this process is expected to be a minor overall loss process from the atmosphere because of the short chemical lifetime and half-life of phenol (Atkinson, 1995).

AB 2588 RISK ASSESSMENT INFORMATION

The Office of Environmental Health Hazard Assessment reviews risk assessments submitted under the Air Toxics “Hot Spots” Program (AB 2588). Of the risk assessments reviewed as of December 1996, for non-cancer health effects, phenol contributed to the total hazard index in 13 of the approximately 107 risk assessments reporting a total chronic hazard index greater than 1 (OEHHA, 1996b).

HEALTH EFFECTS

Probable routes of human exposure to phenol are inhalation, ingestion, and dermal contact.

Non-Cancer: Phenol is a strong eye and respiratory irritant. It is corrosive to the eyes and skin upon direct contact. Acute inhalation exposure may cause nausea, vomiting, cardiac arrhythmias, circulatory collapse, convulsions, and coma. Limited data are available on the chronic effects of phenol in humans from inhalation or oral exposure. In one study, muscle pain, weakness, enlarged liver, and elevated levels of liver enzymes were found in an individual after inhalation and dermal exposure to phenol and other chemicals (U.S. EPA, 1994a).

A chronic non-cancer Reference Exposure Level (REL) of 45 µg/m³ is listed for phenol in the California Air Pollution Control Officers Association Air Toxics “Hot Spots” Program, Revised 1992 Risk Assessment Guidelines. The toxicological endpoints considered for chronic toxicity are the kidney and respiratory system (CAPCOA, 1993). The U.S. EPA has established an oral Reference Dose (RfD) for phenol of 0.6 milligrams per kilogram per day based on reduced fetal body weight in rats, and has not established a Reference Concentration (RfC). The U.S. EPA estimates that consumption of the RfD or less, over a lifetime, would not likely result in the occurrence of chronic non-cancer effects (U.S. EPA, 1994a).

No studies were located regarding adverse developmental or reproductive effects of phenol in humans. Offspring of animals exposed to phenol orally were reported to have reduced fetal body weights, growth retardation, and abnormal development. Decreased maternal weight gain and increased maternal mortality were also observed (U.S. EPA, 1994a).

Cancer: No studies were located on the carcinogenic effects of phenol in humans. In some animal dermal exposure studies, indications that phenol may be a tumor promotor and/or a weak skin carcinogen in mice were observed. The U.S. EPA has classified phenol in Group D: Not classifiable as to human carcinogenicity based on lack of data concerning carcinogenic effects in humans and animals (U.S. EPA, 1994a). The International Agency for Research on Cancer has classified phenol in Group 3: Not classifiable for human carcinogenicity (IARC, 1989a).